

# HSOA Journal of Brain & Neuroscience Research

## **Case Report**

# Postpartum Catastrophe: Cerebral Venous Sinus Thrombosis with Intracranial Hypotension

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### **Abstract**

Postpartum cerebral venous sinus thrombosis with the complication of intracranial hypotension is a rare entity This complication must be kept in mind while evaluating patients with this disorder, as they have predisposing risk factors of hypercoagulable state with spinal anesthesia, so prompt recognition with early diagnosis and treatment is required for a favorable outcome.

#### Introduction

Being a life threatening condition, cerebral venous sinus thrombosis is uncommon. Annual incidence ranges from 1.16 to 2.02 per 100,000 1,2and is more common in females than males, with a female-to-male ratio of 3:13, 4. Common risk factors are pro-thrombotic conditions, either genetic or acquired, obesity, oral contraceptives, pregnancy and the puerperium, malignancy, infection, and head injury and mechanical precipitants.

Cerebral venous sinus thrombosis (CVST) has been associated with intracranial hypotension in postpartum cases, though this connection is relatively rare. After childbirth, women may experience changes in fluid dynamics and hormonal fluctuations that can predispose them to both conditions. In these cases, timely diagnosis is crucial. Magnetic Resonance Imaging and Magnetic Resonance venography can help identify both SIH and CVST. Treatment typically involves addressing the underlying CSF leak, often through epidural blood patches, along with anticoagulation therapy for CVST. Close

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**Citation:** Khalil M, Qadeer J, Naik S, Majid Rajput H, Iqbal M, et al.(2025) Post-partum Catastrophe: Cerebral Venous Sinus Thrombosis with Intracranial Hypotension. J Brain Neuros Res 9: 033.

Received: May 19, 2025; Accepted: May 26, 2025; Published: May 29, 2025

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monitoring is essential to prevent complications and ensure optimal recovery.

Here we are reporting a case of postpartum CVST with Intracranial hypotension.

#### **Presentation**

A female 24 years of age, married having no previous co-morbids presented in emergency with complaint of severe headache for 3 days and numbness of left side of body for 1 day. She had history of Caesarian section at 32 weeks of gestation due to oligo-hydrominos eight days prior to this symptoms onset with outcome of male child. As per her, she experienced headache, sudden onset, bi-temporal, associated with nausea and vomiting, aggravated with standing and relieved with lying. There was blurring of vision in both eyes and buzzing in ears. She also had numbness of left side of body. There was no history neck pain, photophobia, phonophobia, loss of consciousness, fever, sore-throat, ear discharge, fits, or sphincteric incontinence. No history of fall, trauma, use of any contraceptive medication and previous systemic thrombotic events or miscarriages.

On Examination in emergency, she was conscious, well oriented in time, place and person. Her vital signs were blood pressure of 110/70 mmHg, pulse of 86 beats per minute, respiratory rate of 18 breaths per minute, temperature of 98 degree Fahrenheit with Oxygen saturation of 98% at room air. Neurological examination showed Glasgow coma scale [GCS] of 15/15, Pupils were bilateral equal and reactive to light, no neck stiffness and signs of meningeal irritation, Cranial nerves were intact, Power as per MRC grading were normal, Plantar reflex were bilateral withdrawal, Sensation were intact. Spine examination were normal. Dilated fundoscopy was normal. Rest of the systemic examination were unremarkable.

Her preliminary investigations include blood complete picture that showed total leucocyte count of 14760 / microliter, hemoglobin of 12.7 g/dl, and platelet count of 321,000/microliter. Her serum chemistry was normal along with coagulation profile. Hepatitis B and C screening were negative. Electrocardiography (ECG) showed sinus bradycardia. Antinuclear antibodies (ANA) screening was negative. Erythrocyte sedimentation rate (ESR) was 20/mm 1st hr. Figure 1 depict her plain Computed tomography of brain done in emergency on presentation. Magnetic resonance imaging of the brain with contrast and venography done depicted in figures 2 & 3.

Considering the diagnosis of cerebral venous sinus thrombosis and intracranial hypotension, treatment with hydration and anticoagulation started along with conservative management for intracranial hypotension. Anticoagulation therapy with Enoxaparin 1mg/kg subcutaneously 12 hourly was initiated along with painkillers and bed rest. Her symptoms improved markedly on day 7th of admission. She was discharged on novel oral anticoagulant Rivaroxaban 15mg twice per day for 21 days then 20mg once a day for 3 months after opting it in view of postpartum status with advice of good hydration, bed rest and caffeine intake. Follow-up scan was advised after 6 weeks.

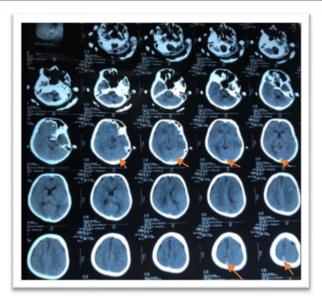


Figure 1: Plain computed tomography brain showed dense clot sign with left transverse cord sign.

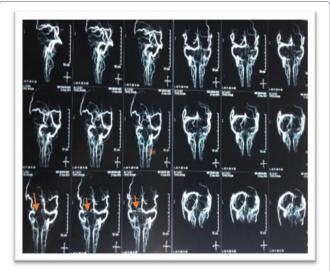
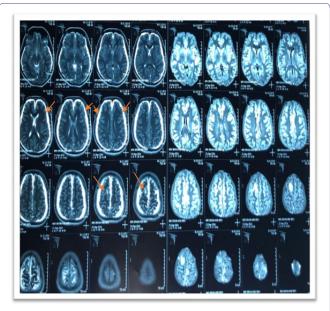


Figure 2: Magnetic resonance venography of brain showed superior sagittal and transverse sinuses are thrombosed along with cortical and cerebral veins

### **Discussion**

Being a rare but important complication, postpartum cerebral venous sinus thrombosis (CVST) with intracranial hypotension is reported. As a clinical and radiological syndrome, intracranial hypotension is caused by spinal leakage of cerebrospinal fluid (CSF) either due to a dural tear, leaking meningeal diverticulum or CSF venous fistula 5. Most commonly presentation is orthostatic headache in approximately 92% of cases 6. The time of headache onset on becoming upright can be anywhere from immediate to many hours later [7,8]. It is reported more frequently in individuals with disorder of connective tissue such as Marfan syndrome, Ehlers-Danlos syndrome, or joint hypermobility syndrome [9].



**Figure 3:** MRI Brain with contrast depicts bright T2 signal area in the right frontal region suggested hemorrhagic infarct. Bilateral diffuse dural enhancement and subdural collections along fronto-temporo-parietal regions were seen.

Postpartum SIH often results from factors like epidural anesthesia, which can lead to CSF leaks. The resultant low CSF pressure may contribute to venous engorgement and stasis, increasing the risk of CVST. Symptoms such as persistent headaches, visual disturbances, or neurological deficits may arise, prompting further investigation. It is a rare cause of CVT occurring in only 2% cases, is characterized by postural headache and a cerebrospinal fluid (CSF) pressure of less than 60 mm of H2O. Although the mechanisms of SIH-induced CVT are not fully understood, three hypotheses have been proposed: [1] Following the logic of the Monro-Kellie doctrine, the loss of cerebrospinal fluid leads to compensatory dilation of veins, which slows down blood flow through the straight sinus, a complication that has been reported in some cases of CVT. [2] Through an epidural incision, CSF flows into the epidural space rather than into the venous system, resulting in increased blood viscosity in the epidural veins 2. [3] The sagging of brain tissues can pull on the parenchymal veins, leading to turbulence or stagnation of the venous blood flow. The most common focal brain injuries of SIH-induced CVT are cerebral venous infarction, cerebral hemorrhage, subarachnoid hemorrhage and focal cerebral edema, and the most common symptoms are epilepsy and limb weakness [4-6].

Recognition and diagnosis of SIH remain challenging because of both imaging and clinical factors. At least 19% of patients with SIH have normal-appearing brain MRI, and brain MRI changes in chronic SIH may disappear over time, despite persistence of the leak 6, 7. Low opening pressure (OP) is diagnostically unreliable as most of the patients have OP within the normal range [10,11]. Even if suspected, localizing spinal CSF leaks and CVF require sophisticated imaging techniques that are not widely available [12]. It is a highly disabling but treatable secondary cause of headache. Can be managed conservatively. If not then with a high-volume non-targeted epidural blood patch. If there is no response to non-targeted epidural blood patches, the site and cause of spinal CSF leak site should be sought

using dynamic CT myelography or digital subtraction myelography, in order to perform targeted treatment with surgery, targeted patching or transvenous embolisation. Several recognized complications of SIH, including subdural hematoma/ hygroma, cerebral venous sinus thrombosis, superficial siderosis, bi-brachial amyotrophy, syringomyelia secondary to pseudo-Chiari malformation, frontotemporal dementia-like syndrome due to brain sagging and coma.

In one of review of 563 patients with clinical and/or imaging findings of SIH, 0.9% of patients were diagnosed with CVST and 3 of these patients (60%) had additional severe complications [13]. These case reports highlight the importance of prompt diagnosis and treatment. We are reporting this case as the combination of both entities need to be kept in mind while evaluating headache in postpartum patient and must be managed early to avoid catastrophy.

#### **Conclusion**

Postpartum CVST with Intracranial hypotension is a rare entity and must be kept in mind to ensure early recognition and prompt action for better management and prevention of complications to reduce morbidity and mortality.

#### **Declaration**

**Ethical approval:** It has been approved by Departmental Review Board and Ethics Committee, Neurology Department of Pakistan Institute of Medical Sciences.

**Consent to participate and publication:** Consent to participate and publication has been taken by participant.

**Competing/conflict of interest:** The authors declare that they have no competing interests.

Funding: No funding sources.

Availability of data and materials: Not Applicable.

**Author's contribution:** All authors participated in data collection, manuscript writing. All authors read and approved manuscript.

**Acknowledgement:** I am really grateful to my co-author who contributed in this study.

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